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CURRENT THERAPY

Acute Myocardial Infarction

(continued)

Bed Rest Vs. "Arm-Chair Treatment"

By Jack A. Bernard, M. D., *El Paso*

With a Discussion by Dr. John E. Morrison, El Paso

Since the 1920's the classical treatment for myocardial infarction has been absolute bed rest for three weeks—six weeks—or even more. About 1937 Dr. Samuel Levine noted improvement followed by prompt recovery in a coronary patient who was moribund with severe congestive heart failure and pulmonary edema, when this patient was placed in a chair after all other measures had failed, in the hope to divert fluid from the lungs and the lower extremities. Since that time he and his colleagues have demonstrated the beneficial effects of this method of treatment, referring to it as the "arm-chair treatment."

Statistics Favor Arm-Chair Treatment

It has been pointed out that long bed rest is actually harmful. Wood reported "arm-chair therapy" reduced the mortality by 50 per cent. Helander also showed that the death rate was doubled by the conventional method of treatment. In Levine's cases, the incidence of complications was decidedly less among the chair-treated patients. In view of these statistics it is surprising that this type of treatment is not more generally accepted, since it has been now 20 years since it was first described and since subsequent reports have confirmed its value.

Purpose of Rest

The purpose of bed rest is to rest the heart but it has been shown that cardiac work is actually increased in the recumbency position, due to an increase in circulating blood volume due to the maximal venous return from the periphery. The recumbent position also encourages the pooling of fluid in the pulmonary circuit. Wood found cardiac work less in the sitting position. Catherization studies by Coe showed the work of the heart 23 percent less when the person is in a chair than when the person is in bed. It seems evident, therefore, that the work of the heart is decreased by chair treatment.

There are many clinical observations also that cause one to question the need for strict bed rest. Many patients have been observed to have had "silent coronaries" and have done quite well without bed rest. Many patients have been treated for acute coronary occlusion that have done quite well in spite of activity.

Harmful Effects of Bed Rest

Finally, both Masters and Levine point out that "strict bed rest produces anxiety and depression, loss of muscular and vascular tone, constipation and distention and predisposes to venous thrombosis." Thus Master writes "chair treatment reduces all these ill effects and lessens the need for prolonged anticoagulant therapy." Anxiety is harmful for another reason, in that studies have shown that emotion causes a marked increase in cardiac output.

In view of the above it is evident that patients are kept in bed too long and the period of bed rest should be shortened. The severely ill patient

in shock should be kept in bed until the usual therapeutic measures have brought this under control. The patient with congestive failure is certainly benefited by approximating the sitting position or by placing in the arm-chair as recommended. Many patients with angina are benefited by the upright position.

Levine advocates the arm-chair treatment for all cases of acute coronary thrombosis from the onset of the attack and particularly for patients with congestive failure. In most cases a patient may be out of bed in the first two days. He remains in the chair as long as he is comfortable. He is helped by two people, one on each side, being careful not to put any pressure on the back of the patient's knees or calves in order not to interfere with the circulation of the lower extremities. The patient is usually allowed to feed himself, comb his hair, but he is not allowed to shave himself.

Contraindications to Treatment

Contraindications to the arm-chair treatment include severe shock, extreme debility and weakness, or a cerebrovascular accident. It is felt by the chair advocates that high fever, severe pain, a need for oxygen, cardiac irregularities do not prohibit the use of the chair.

They *emphasize* that the arm-chair treatment is *not* meant to allow the patient more physical activity. The patient is *not* allowed to walk in less than three weeks following the onset of his attack. Levine points out from pathological studies that rest should be advised for four to six weeks, at the end of which time final healing and scar formation are thought to be complete.

Caution

It is pointed out that one of the hazards of "chair treatment" is that it may encourage an easy going attitude toward the gravity of the process being treated. It is emphasized that the patient should be kept as quiet in the chair as he would in bed. This is repeatedly emphasized: *that rest in the chair should be carried out with the same strict adherence as rest in the bed.*

Harmful Effects

There have been no harmful effects noted from the chair treatment. Observations made on patients with acute myocardial infarction when the patients were got out of bed into a comfortable chair showed that there was no change in pulse and respiration. A very slight dimininution in blood pressure occurred, suggesting cardiac work load may have been decreased by chair treatment.

Follow-up studies have shown no complications attributable to this type of treatment. There is no evidence that mortality is increased. Follow-up roentgenologic studies have shown no instance of ventricular aneurysm. No deleterious long range effects have been noted.

Summary

"Arm-chair therapy" seems superior to the traditional strict bed rest regimen. Physiologic as well as clinical studies support the view that cardiac work is increased by recumbency. Mortality is not increased by the chair treatment. No complications attributable to this treatment have been noted. Contraindications include shock, weakness and cerebrovascular accidents. It is emphasized repeatedly that the treatment does not allow the patient more physical activity.

Discussion by Dr. Morrison

Dr. Bernard has given us an excellent review of the subject of armchair treatment of myocardial infarction. Probably in the near future this type of management of these pateients will become as routine as bed rest has been in the past, at least with patients under close supervision. Certainly it offers great relief to some patients. The dramatic improvement in acute pulmonary edema achieved by getting the patient out of bed and into a chair is familiar to every one. There are also many patients with coronary insufficiency, without pulmonary edema, who experience their greatest difficulty in the recumbent position.

There are certain drawbacks in trying to apply this type of management to all cases of myocardial infarction. Most patients with mild or moderately severe attacks are cared for without the aid of special nurses. In many areas there is a dire shortage of floor nurses and trained attendants. The effort of geting in and out of a high bed without skilled attendants (Levine advises one on either side of patient), together with the possibility of a patient being left in a chair for some time after becoming tired, would negate some of the advantages of this type of treatment. It is a little frightening to me to think of leaving a patient with recent myocardial infarction with elevated temperature, a rising sedimentation rate and a cardiogram that has not yet stabilized, sitting in a chair for variable and unpredictable periods of time.

Dr. Bernard has presented the case for armchair treatment of myocardial infarction well indeed. I would like to congratulate him on his concise and highly instructive presentation of this very provocative subject.

By W. Compere Basom, M. D., El Paso, Orthopaedic Editor of Southwestern Medicine

ORTHOPAEDIC SURGERY NOTES

Dr. Louis W. Breck of El Paso has submitted his impression of the Third Annual Automotive Crash and Field Demonstration Conference which was held at the Holloman Air Force Base, New Mexico, Nov. 12-13, 1957 under the direction of Col. John Paul Stapp, MC.

Dr. Breck reported that Col. Stapp was an excellent master of ceremonies and that his staff put on an outstanding program. Commanding General L. I. Davis gave an address of welcome and showed a movie on guided missiles which gave a good idea of the set up and operations at the base. There is a ground test track there 35,000 feet long and absolutely straight and flat. It is the biggest thing of this kind anywhere in the world.

Much Controversy

On the question of seat belts, since there is so much controversy, your reporter would like to emphasize the fact that seat belts are important. A young medical officer in the Air Force demonstrated that he could withstand 26 G. with a lap belt only and he was obviously not hurt at all. With the same track and the same force (26 G.) a dummy was demonstrated. The dummy was thrown about 15 feet. This force would have completely demolished a human. The dummy did not have a seat belt. In other words all of us should use our seat belts and should have them available. It is too late to put the seat belt on by the time the accident occurs.

Colonel Stapp had three types of deceleration equipment. It was noted that 26 to 30 G. is about the most that human experiments will tolerate with only a seat belt in testing demonstrations of deceleration. One of the types of deceleration equipment is a bopper test track which is spring operated and is decelerated with very carefully made lead brakes on brake shoes. Another is a simple swing seat which is self descriptive.

The third and best one is called the Daisy track which is to be actuated eventually with compressed air. Colonel Stapp is now using catapult explosive charges. This is very carefully made and controllable track. A bear was decelerated at 25 G. The bear was not hurt at all. Therefore it is obvious that a lap belt or seat belt is most helpful in decelerations. Apparently the human pelvis offers the best protection for seat belt retentions of any animal known, and in fact we seem to be especially prepared to use seat belts effectively.

In connection with Biostatistics of accidents in the Cornell University Accident Survey, Mr. Tracey gave an excellent presentation. He is in charge of field work and of encouraging police officers particularly and doctors and others secondarily into doing the best they can on reporting accidents accurately.

The survey now has quite a large series, but wants to run the series up to 20,000 or 25,000 before making a more detailed report. The survey has now expanded the project with considerably more financial aid and expects to get 10,000 good reports a year. These extensive statistics are needed because from 1956 on there are many more accidents reported in which the victims have seat belts. However, the survey is interested in all types of statistics on accidents. It is now working in 75 areas in 16 states including Texas.

Arizona was an early state to be included, and probably West Texas will not be included because it so resembles Arizona where there are extensive statistics. Mr. Tracey said that everyone was quite cooperative in gathering these statistics. Over 90 per cent of the forms on accidents get completed satisfactorily.

Main Approach

The main statistical approach is to use random sampling from all over the country. An interesting thing is that early cars and later cars up to 1956 showed approximately the same statistics in regard to incidence of injuries and severity of injuries.

One thing stands out above everything else and that is that ejection is the worst thing that can possibly happen and the worst thing statistically as a cause of serious injury and death. One other serious thing that can happen is to be hit directly from the side with a vehicle going at high speed and with the impact occurring right where the passenger is sitting.

There is very little that can be done about this particular type of situation except to try and not get hit directly on the side. As the speed goes up in all accidents, there is a marked increase in the injury rate. Sixty miles an hour seems to be a good ranging point.

In accidents below 60 and above 60, the rate is three times as great above 60, both as to injury rate and mortality. However, the survey stresses that speed, although it is very important, is not

necessarily the major factor in regard to decreasing automobile injuries and deaths. As far as the 60 mile an hour speed is concerned, a practical angle of this is that if an impending traffic situation develops and you are the driver, get down well below 60 as soon as possible before getting involved in any type of collision.

As an aside in this discussion, it was apparent to everyone in the conference and was brought out repeatedly by different discussers and assayists that there is a certain group of accidents called non-survivable and that analysis and attempting to improve the statistics in these is hardly worthwhile on an economic or practical basis.

Non-Survivable Group

There is not going to be much time or energy spent on the non-survivable group. Examples of non-survivable accidents are roll over at 90 miles an hour or striking a total obstruction such as a tunnel side at 30 miles an hour. Both of these develop 50 G. or more in the occupant, and he would be extremely lucky to survive either of them. Now in regard to seat belts and estimates as to their effectiveness by the Cornell people: A seat belt may be anywhere from 30 per cent to 60 per cent effective.

They believe they are probably 60 per cent effective at reducing mortality only. They are willing to state positively that they are at least 30 per cent effective and probably 45 is a good average as to their effectiveness on conservative estimates.

In their analysis of 700 seat belt cases, there has not been a single death in any case except in the non-survivable group where the patient couldn't possibly have survived anyway.

In other words, their small series of 700 seat belt cases, showed total effectiveness of seat belts so far. There were no belt failures in these 700 cases and there was not a single death caused by a belt. It is quite evident that there may be a freak case of someone being killed by a belt but these cases must be extremely rare and certainly under one per cent of seat belt accidents.

In the Cornell report there were no more injuries with high powered cars than with low powered cars. It is just the same, in old cars and in 57's. There is no correlation between horse power and speed in their statistics. Heavy and light American cars are equally dangerous from the statistical standpoint.

It is obvious that in a collision between a very heavy and a very light car, one would be better off in the heavy car but statistically there has been no difference in the number or severity of injuries sustained in heavy or light American cars. They do not have data on foreign cars because they have had so few accidents in which they were involved that the data are insignificant.

Dr. James J. Ryan, professor of engineering at the University of Minnesota, has been working on safety cars and safety bumpers for a long time. He has a safety bumper that is highly efficient. It is essentially a shock absorber type of device with big shock absorbers in the bumper supports on each side. There are several versions of this. A heavy one for trucks and lighter one for cars. Dr. Ryan also has a steering wheel that bends over and gets out of the way. This is pulled over by a cable so the occupant can't strike the wheel.

A seat belt was demonstrated and an ordinary nylon belt was tested up to 6,600 pounds without breaking anywhere. The fastening of the nylon to the buckle is important. You should use good, big washers to bolt the belts to the automobile floor. A flat statement was made that a 6,000 pound belt is totally effective in an automobile wreck unless you hit your head or are in a non-survivable accident.

In moving pictures shown, 15 G. causes the subject to go away from the seat and into the seat belt. Subjects who have been subjected to this usually have muscle pain in their upper abdomens for four to 24 days but nothing serious. Twenty seven G. for 1/20th of a second gives spine pain in the upper back and all of the subjects who were subjected to 27 G. felt that this was absolutely all they could take.

Six G. will expel the occupant from the car very easily. Twenty seven G. gives a 4,700 pound pull on the seat belt.

If you are going 30 miles an hour and go into a total obstruction with instantaneous deceleration, you develop 40 G. and are right at the edge of a non-survivable accident. With a seat belt you would probably survive but you couldn't count on it as 40 G. is at the reasonable limit of survival.

Recommendations

The S.A.E. Seat Belt Committee discussion revealed that 3,000 pound standard for seat belt with the top of about 4,000 pounds was all that would be recommended because of the relatively small increase in safety in the higher bracket.

Dr. Duncan McKeever brought out a new theory about whiplash injury in that it actually is not a whiplash but a forward thrust of the body and cervical vertebrae against the skull. There are some interesting forces to figure from a standpoint of physics which brings us around to this writer's opinion, and that is that the diagnosis of "whiplash injury" is not accurate and actually we should designate this as "sprains of the cervical spine" or "strain of the neck ligaments and muscles" since whiplash is merely the mode of acquiring these.

A tightly fastened seat belt and a straight up sitting position are the best preventatives of severe injury from rear end collisions in the automobile.

THE PRESIDENT'S COLUMN

The Author Outlines His Position

By Louis G. Jekel, M.D., *Phoenix*

In previous months we have discussed the Southwestern Medical Association, dwelling on its organizational structure, and its purposes and objectives. It was brought out that there exists a set of officers. One of these officers is the President.

Dr. Louis G. Jekel

I wish at this time and during the next several months to express my views as to the duties, obligations, rights, and privileges of the President, and to outline a program which, it is hoped, could serve as a model for future years.

It has been customary for the President to write each month for *Southwestern Medicine* a column known as the President's Page. Many medical journals have such a regular feature. The President is in a good position regarding this article, for he may write anything on any subject and almost at any length. He has a sounding board for his views. He need only stay within the bounds of decency and propriety. And, there being no rebuttal, no one can argue with him.

The reader is in a somewhat less favorable position. Here can be published the good or bad opinions of one man. The material may be well written or poorly written. It may be important or unimportant. It may be informative or instructive, or not. It may be completely worthless, and often is. Your only defense, dear reader—and this may be well-nigh life-saving—is that you do not have to read it.

Friendly Critic

A well-meaning, friendly critic has pointed out that the President's Column would better be left unwritten unless the President really has something to say. I agree absolutely, although by doing so I may amputate the greater part of my literary career. In self-defense, then, I must include this column and your courtesy in reading it among the privileges accorded me as holder of this office.

In return for your courtesy I shall endeavor to produce something worthwhile in the sense that you shall not, having read it, feel that you have wasted your time.

My material will more than likely be along the lines of the history and organization of the Southwestern Medical Association, the History of Medicine, especially as it pertains to the Southwestern United States, the opportunities presented in the region for clinical research and medical writing, some phases of the practice of medicine, and perhaps some amusing anecdotes.

I hope and pray that whatever I write or say may work for the benefit of our organization and to the satisfaction of its members.

Social Security Footnotes

To support socialistic practices in regard to retirement funds requires that, for the sake of consistency, socialized medicine also be supported. If one believes that the federal government should tax everyone to provide an income for each upon retirement or disability, one must also believe that the same government should, with equal propriety, tax everyone to provide medical care for all!

Edited By ANDREW M. BABEY, M.D., LAS CRUCES, N. M.

APHORISMS and MEMORABILIA

Truths and Concepts Concerning the Bronchopulmonary System

(continued)

15. "In every case of tuberculous pleurisy with effusion look for future seeding, for many such cases represent hematogenous disseminations."—J. B. AMBERSON, JR., *loc. cit.*

16. "One should palpate tuberculous cervical glands with great care to avoid reaction."—J. B. AMBERSON, JR., *loc. cit.*

17. "Look for a cavity somewhere in the lung when you find a positive sputum."—J. B. AMBERSON, JR., *loc. cit.*

18. "There is a real connection between the mediastinal lymphatics and the supraclavicular nodes, but none between the mediastinal and cervical nodes. This explains the occasional occurrence of a Virchow's node in carcinoma of the lung."—J. B. AMBERSON, JR., *loc. cit.*

19. "Every case of lung abscess should be bronchoscoped."—J. B. AMBERSON, JR., *loc. cit.*

20. "It is not uncommon for a pleurisy with effusion to start exactly like a pneumonia with a chil, fever (up to 105) and stitch in the side."—J. B. AMBERSON, JR., *loc. cit.*

Crepitations

21. "Pleural crepitations heard above the level of a resolving pleural effusion may at times deceive you into thinking you have parenchymal involvement."—J. B. AMBERSON, JR., *loc. cit.*

22. "Recurrent unilateral or bilateral spontaneous pneumothorax is very commonly due to rupture of superficial emphysematous blebs."—J. B. AMBERSON, JR., *loc. cit.*

23. "If you get musty or odorous pus and a culture is negative, don't be deceived. Do an anerobic culture."—J. B. AMBERSON, JR., *loc. cit.*

24. "If you find lung abscess in a man over 50, think of carcinoma of the bronchus as the cause."—J. B. AMBERSON, JR., *loc. cit.*

25. "Bronchiectesis is almost never diagnosed by bronchoscopy."—J. B. AMBERSON, JR., *loc. cit.*

26. "Congenital cystic disease of the lungs is more mystic than cystic."—(Quoted by J. B. AMBERSON, JR., after GRETHMANN), *loc. cit.*

27. "The more fibrotic and emphysematous a lung, the more atypical will the pneumonia be."—J. B. AMBERSON, JR., *loc. cit.*

28. "Every chest infection damages the lungs."—J. B. AMBERSON, JR., *loc. cit.*

29. "Before operating on anyone with much laryngeal involvement, take a chest plate to be sure there is no aspiration pneumonia."—J. A. AMBERSON, JR., *loc. cit.*

30. "Fungus disease of the lung usually turns out to be tuberculosis, and frequently atypical hematogenous tuberculosis, for fungus disease is uncommon."—J. B. AMBERSON, JR., *loc. cit.*

31. "In any pyrexia of long duration, look closely at the mediastinum for tuberculous glands."—J. B. AMBERSON, JR., *loc. cit.*

32. "When acute disease of the lungs occurs in persons with rickety deformity, the violence of the symptoms is often quite disproportionate to the extent and severity of the pulmonary disease, and may thereby suggest unnecessarily active treatment."—THOMAS ADDISON'S WORKS, *New Syndenham Soc.*, London, 1868 vol. 36, page 65.

33. "We may be called to see a case of pleurisy before a single physical sign has developed. In such a case it may be doubtful whether the pain arises from pleurisy, rheumatism, neuralgia, or the approach of shingles."—THOMAS ADDISON'S WORKS, *loc. cit.*

34. "Sound is transmitted through bodies by a successive motion of its molecules, the whole mass not necessarily undergoing any change of place, whilst the movement appreciable by the hand is the change of place which an entire body undergoes."—THOMAS ADDISON'S WORKS, *loc cit.*

35. "I shall content myself with offering a caution not to mistake the crepitations so often attendant upon recent pleuritic effusion for the crepitatory and mucous rales of bronchopneumonia."—THOMAS ADDISON'S WORKS, *loc. cit.*

36. "The reason that most metastatic pulmonary malignancies are shown as rounded, sharply defined opaque shadows in the roentgenogram is due to the large number of end arteries in the lungs and to the yielding nature of lung parenchyma. A malignant cell lodges in an end artery and multiplies; as the resistance in all directions is the same it grows as a round, discrete tumour."—RADIOLOGIC MAXIMS, *Mississippi Valley Med. Jour.*, 61: 114, 1939.

ORIGINAL ARTICLES

The Care of Multiple Injuries*

By Preston A. Wade, M.D., *New York*

The problem of the seriously injured patient has never assumed as great an importance as it has in the present generation. In this modern day of rapid transportation, as a result of the combination of excellent highways and high powered motor cars, there is an increasing number of civilian casualties, so that even the smallest hospitals are continually faced with the problem of the treatment of patients with multiple injuries.

On an ordinary holiday weekend three or four hundred fatalities may be expected and, in addition, thousands of serious casualties which cause long, painful and costly disabilities. It is well known that the number of civilian casualties arising from motor car injuries in one year is considerably greater than the combined number of casualties suffered during all the wars in which the United States has been involved.

On the other hand, the ominous threat of an atomic war makes us realize that in another conflict most of the casualties will be in our cities, will be civilian casualties, and will need to be taken care of by our present medical facilities.

Even though the threat of war may be more remote than it was a few years ago when civilian defense plans were first initiated, it is essential that we keep in mind these facts and be prepared for catastrophes in every community.

Although most hospitals have a plan on paper for disasters, when faced with an unexpected catastrophe they may find that the plans fall short, and it is from the actual experience of hospitals in catastrophes that we may learn important lessons.

It is interesting to read the report of the three tornados which occurred several years ago—one in Waco, Texas; one in Worcester, Mass.; and one in Flint, Michigan. These were very similar in many respects, in that each had about the same number of fatalities and each about the same number of casualties, and in every case the difficulties encountered seemed to parallel one another. In Waco there were 113 deaths and in Worcester 114 deaths.

In one there were 1500 casualties and in another 950 casualties. In each instance the failure of proper organization caused some of the most serious difficulties. The necessity of preparedness and organization are the two factors stressed in each of the reports to control the fear, panic, confusion, and chaos which resulted in each instance. The question of proper communication and proper transportation seemed to be stumbling blocks in all of these disasters.

In the Worcester report, particularly, the writer was impressed by the difficulty in receiving patients properly and in sorting the injured in hospitals, so that the seriously injured received prompt and efficient care.

In all of these cases the medical services seemed to be adequate and the supplies were adequate, but there was a question of faster communication to the personnel needed and transportation to get them to the hospital. In all of these the emphasis was on a well-trained and well-organized small group, rather than a large group of poorly organized and poorly trained personnel.

Another lesson learned in the disasters mentioned above was the high infection rate of the sutured wounds. This is a lesson which must be emphasized to the young surgeon, who is accustomed to laborious suturing of open wounds in civilian practice.

First Aid

In many instances of automobile crash injuries, as well as what may be expected in an atomic war, most of the cases will receive first aid from a layman and untrained personnel. The Red Cross is doing a very good job in attempting to train as many first aid instructors as possible, and it is certainly the duty of the medical profession to educate as many as possible of the lay public in the fundamentals of first aid.

It also becomes necessary now for the medical profession as a whole to take an interest in the problem and see to it that all physicians, even though they may not be interested primarily in trauma, have some basic knowledge of first aid in

*Presented at annual meeting of Texas Orthopaedic Association, Dallas, April 29, 1957.

order to help train personnel when the occasion arises.

There are several general principles in first aid which I should like to mention. The first is the priority of conditions that need first aid treatment.

There are three conditions in which first aid therapy may be of considerable help in saving lives.

The first is the treatment of obstruction to respiration—the maintenance of a free air way; the second, the stoppage of hemorrhage; and the third, the treatment of shock.

Certainly, even the lay person realizes that without the free air exchange the patient cannot survive, and when possible, mucous, blood, and vomitus should be removed from the mouth and pharynx in order to prevent aspiration and suffocation.

Injuries about the mouth and jaw sometimes necessitate traction on the tongue and even an emergency tracheostomy may be necessary to assure an open airway.

The second condition in which first aid treatment is essential to save life is the stoppage of hemorrhage. There has been a great misconception as to the means of stopping hemorrhage, particularly in the early years of World War II when first aid students were advised to use pressure points and tourniquets, particularly in Red Cross courses and in most first aid manuals.

There is one efficient, harmless and easy means of stopping hemorrhage, and that is by the pressure bandage. The indiscriminate use of the tourniquet is certainly to be condemned.

As a matter of fact, Sir Reginald Watson-Jones has indicated that in his opinion the tourniquet should not be permitted in first aid kits, so that the person giving first aid would not be tempted to use it.

Life Saving Measure

There is no doubt that on occasion a tourniquet may be a life saving measure, but such an instance is quite unusual, and the tourniquet must necessarily be applied immediately after the accident. In most cases, the complete severance of an artery, such as a complete amputation of a limb by a railroad train will result in a spasm of the artery preventing fatal hemorrhage.

If the artery is only partially divided, this spasm does not prevent continuing hemorrhage, however, and in these rare cases a tourniquet may be necessary. However there is danger in a tourniquet applied too loosely; venous bleeding may be serious or fatal.

A tourniquet applied too tightly causes damage to nerves and blood vessels and deprives the limb of collateral circulation, and if left on too long, will result in ischemia with tragic results.

In my opinion, the teaching of students in first aid pressure points, and in the use of a tourniquet is an entirely useless procedure and should be discontinued, except to stress the dangers of their application. It should be emphasized that the one means of stopping severe hemorrhage is by means of a pressure bandage directly over the bleeding area.

The third important condition in which first aid treatment is helpful is the condition of shock. It is seldom possible to treat shock properly at the scene of the accident, but certain measures can be taken to prevent these conditions which cause shock.

The stoppage of hemorrhage, the proper splinting of a fracture, and the application of blankets to keep the patient warm are important steps in every injured case. On occasions, intravenous fluids may be given immediately by the first aid attendant, if facilities are available.

It is interesting to note, however, that in the Worcester tornado report, the reporter states that in a large percentage of those cases having received intravenous fluid on the field, the needles had slipped out and the fluid had infiltrated the skin.

He gave the impression that perhaps in many of these cases intravenous therapy had merely caused delay in the transportation of the patient to the hospital and had not served a useful purpose.

Perhaps it would be wise to make sure that intravenous therapy be efficient before efforts are made to institute it in the field.

Another important consideration has to do with the splinting of fractures and the protection of the patient in transportation. All bones and bones suspected of being fractured should be splinted before the patient is transported, and the simplest type of splinting is the best.

In the upper extremity, the upper arm may be strapped to the body for support and a board splint or magazine applied to the forearm for immobilization.

Fractures of the thigh and hip and many fractures of the lower leg should be splinted by means of a Thomas splint with moderate traction.

This means of transportation prevents considerable damage from motion at the fracture site. The lower leg may be splinted in a pillow board splint, if a Thomas splint is not available.

One of the greatest contributions made by the Trauma Committee of the American College of Surgeons has been the work done on the teaching of transportation of the injured.

The emphasis on the splinting of fractures at the site of the accident, as indicated by the aphorism, "splint 'em where they lie", has done a great deal to teach the medical profession the necessity of early splinting of fractures. Not only

does splinting protect the fractured bone fragments and adjacent soft parts from further injury, but it is also helpful in preventing blood loss and alleviating pain. Proper splinting of fractures is one of the most important steps in the prevention and treatment of shock.

It may be mentioned that other first aid measures in the transportation of the injured has to do with the injuries to the neck in which, of course after examination for spianl cord injury, the patient is transported on a flat surface. Great care is to be taken to prevent flexion of the cervical spine.

The patient with a suspected injury to his back should be examined for evidence of neurological involvement and should of course be transported on a board face up or on a blanket face down, so as to cause no flexion of the spine and to prevent damage to the spinal cord.

Dangerous Illustration

One of the first measures often taken by well-meaning by-standers is to lift the head of the patient and offer a glass of water or some stimulant. In recent issues of first aid manuals, there are illustrations of "the proper way to give an injured patient a drink." This is a dangerous illustration and should be deleted from the manuals.

No injured patient should ever be given a drink of a liquid. In the first place, he cannot absorb the fluid, and it will interfere with the administration of anesthetic later, and if the fluid be a stimulant, such as brandy or whiskey, the patient may be accused of intoxication when he arrives at the hospital for treatment.

The result of the treatment of a patient with multiple injuries may be determined by the treatment he received in the accident room. If on admission, the patient is examined by a competent observer and a proper course of treatment instituted, many serious disabilities may be avoided and many fatalities prevented.

A patient entering the hospital with a single injury, such as a fracture of the shaft of the femur or a fractured skull, will usually be assigned immediately to a special department, and his care will be directed by one surgeon who is responsible for his treatment.

There is usually no problem in assigning the responsibility in the case of a single injury.

However, if the patient enters the hospital with a combination of various injuries, each of which may be in the domain of a very specialized department, it may be quite difficult to be assured that the patient will receive proper care because of the divided responsibility.

This failure to coordinate several surgical specialties in the severely injured patients is too often the cause of neglect in many hospitals.

In most instances the first individual to examine the seriously injured patient is the least experienced member of the staff. He may be an intern or a junior resident, or a substitute medical student. He must then decide who should be called to direct the care of the patient.

In many cases the assignment of the case does not constitute a problem if the most severe injury is obvious and is confined to one speciality.

However, the problem may be a very serious one, if, for example, the patient has a neurological condition which needs attention and an abdominal injury in addition to multiple fractures.

In such a case, one surgeon should be assigned to take charge of the case, and he must take the responsibility for the care of the patient regardless of what serious injuries involve other surgical specialties.

It is not important whether the surgeon in charge be the neurological surgeon, general surgeon, chest surgeon or orthopedic surgeon, but it is essential that whoever is responsible assume his responsibility and see to it that the patient is not neglected because of the lack of coordinated care.

In many hospitals the hospital itself assigns the direction of the treatment of the severely injured patient to one individual, and it is his responsibility to see to it that other consultants are called and perform their functions at the proper time. It is most important that the responsible surgeon see the patient immediately, examine him carefully, and direct his immediate treatment. Telephone consultation and direction of the case without examination often leads to disaster. Too often, particularly in the late hours of the night, the responsible surgeon will take the diagnosis and observation of an untrained or inexperienced intern and neglect his most important duty, which is the complete and thorough examination of the patient as soon as possible after his admission.

When a patient is admitted to the accident room, the first duty of the surgeon is to make a complete and thorough examination, removing all of the clothes from the patient and examining every part of the body. The most obvious serious conditions must be considered first.

An open airway and satisfactory respiratory exchange is essential for survival, and all mucous, blood, and vomitus must be removed from the mouth and pharynx, sucked from the bronchial passages, and if necessary, a tracheostomy performed, particularly if there is paradoxical respiration from a crush injury to the chest.

Sucking wounds of the chest must be controlled and hemo- and/or pneumothorax should be evaluated and adequate decompression carried out.

The next most important consideration is the

treatment of hemorrhage and the treatment and prevention of shock. Every case that arrives at the hospital after serious injury must be treated as a good candidate for shock, if he is not in shock at the time of admission.

He must be observed for internal hemorrhage. He must immediately have blood drawn for grouping and cross-matching, and sufficinet blood should be made available when it is necessary.

External hemorrhage should be controlled by means of pressure bandages, as mentioned above.

It is rarely necessary to apply a tourniquet in the accident room. It is often necessary to remove a tourniquet if the bleeding continues due to the fact that the tourniquet has been applied too loosely causing a continuation of venous bleeding.

If a tourniquet has been applied tightly to an injured extremity, and the bleeding has ceased, it is better to leave it in place until proper preparation is made for the treatment of shock and its prevention, because sudden removal of a tourniquet from an injured limb may suddenly send the patient into shock. The patient should be left in the emergency room until such a time as his condition is stabilized.

X-rays should be taken in the accident room by means of a portable machine, so that the patient does not have to stand the transfer from accident room to x-ray and return.

If it is necessary that the patient be operated upon immediately, he should be sent to the operating room where he can be observed, prepared, and the proper time chosen for operation.

He should not be sent to his ward and then be transferred again to the operating room. To transfer an injured patient from stretcher to bed and bed to stretcher is harmful.

The indiscriminate use of morphine in shock is to be condemned. Morphine should be given only for pain and not routinely for the treatment of shock. The danger in morphine administration is that it may not be absorbed when the patient is in shock, and a second dose may be given later, and when the peripheral circulation picks up the multiple doses of morphine, dangerous poisoning may result.

If the patient has a fracture, or if a bone is suspected of being fractured, and no splints have been applied before being transferred to the hospital, proper immobilization must be instituted in the accident room. If the immobilization is not properly applied, this must be corrected in the accident room.

It may be pointed out that while the examination and correction of the serious conditions of interference of respiration, shock, and hemorrhage are being taken care of, blood is drawn for grouping and cross-matching, and tetanus antitoxin or a booster dose of tetanus toxoid is administered in the case of open wounds. The initial doses of a broad spectrum of antibiotics are also given in the accident room.

After the emergency and urgent problems have been evaluated, it then becomes necessary to work out a plan of priority for the treatment of other injuries. The patient must be evaluated as to every possible area in which an injury may be sustained.

We have found in the statistics of the Auto-Crash Injury Research program at Cornell and verified by the admissions to our hospital that there is a very definite pattern of frequency of injury to various areas of the body.

The head was found to be most frequently injured, and these injuries could usually be ascertained or suspected because of the external evidence of injury to the head as well as the state of consciousness.

The evaluation of the head injury is an important factor in determining whether or not the patient can be expected to withstand operative procedures for injuries to other systems. It is most important that this evaluation be determined within as short a time as possible after admission to the hospital.

The next area most frequently involved in injuries in automobile accidents was that of the lower extremities. Here again the injuries to the lower leg and thigh were quite obvious if only a single area was involved, but the injury to the hip in particular was often masked by the other lower extremity injuries and even an x-ray examination, often inadequate in the emergency examination, would fail to disclose the posterior dislocation with fractures of the posterior lip of the acetabulum.

The third area most frequently injured was that of the chest and dorsal spine. As mentioned above, injuries to the chest were often obvious due to the interference with respiration; and crushing injuries to the chest often necessitate a tracheostomy.

This procedure has been used much more frequently in recent years even in multiple fractures of the ribs without actual interference with the physiology of respiration.

Certainly in paradoxical respiration, it is often a life saving measure, and this procedure alone may be enough to take care of the interference with the respiration and obviate the necessity of traction to the chest wall or internal fixation of the segmental fractures of the ribs.

The tracheostomy, although a simple operation, has certain complications and should never be done as an emergency per se but should be done in the operating room with careful preparation and with careful technique.

There are certain precautions which should be

taken, particularly in the use of a long enough tube with a large enough diameter so that full benefit of the airway may be obtained. A transverse incision is to be preferred over the vertical incision because the latter leaves a disfiguring scar.

One of the frequently missed injuries in this area, we found to be the fracture of the dorsal spine because in the patient having multiple injuries, this area may not have important symptoms, and unless the injury is suspected and proper x-rays taken, serious damage may be done to the spinal cord by improper movement of the injured patient.

The next area found to be injured most frequently was that of the upper extremities, and often the injuries were of a minor nature and missed for several days because of the more serious injuries elsewhere.

Injuries to the pelvis and bladder were of less frequency than those mentioned but often were not discovered because of inadequate investigations. If an injury to the bladder is suspected, immediate catherization should be performed if possible, and if the catherization is difficult, injury to the urethra may be suspected or discovered, and proper steps taken to treat the injured urethra.

X-ray examination of the bladder and kidneys may be done, and considerable information gathered from the catherized specimen. Abdominal injuries occurred less often, but they must always be suspected.

They are often masked and the symptoms may develop hours after admission to the hospital. Frequent observations of the suspected injured abdomen should be made, and although there is considerable difference of opinion as to the efficacy of the abdominal tap, it is, when positive, very helpful in making a diagnosis of intra-abdominal hemorrhage.

Even though the patient may have multiple injuries of a serious nature, it is essential that the fracture be treated as soon as possible, and if one of the injuries is an open fracture, the surgeon should be alert to the fact that early operation is essential, and he must seize the first opportunity to take care of the fracture so as to prevent loss of a limb or late disability.

Too often the patient with a suspected head injury or an abdominal injury may be allowed to lie in bed for days before proper disposition is made of an obvious open fracture that should be treated immediately. This, of course, is a question of surgical judgment, and the patient's life should not be endangered by an procedure which can be delayed.

However, often times a debridement can be done in the presence of a head injury and the anesthesia can be local, and even if general, it is not necessarily contra-indicated in a head injury. Cooperation between specialists and sympathetic understanding of the problems of multiple injuries is necessary in any hospital where large numbers of seriously injured patients are received.

Fortunately, it is relatively uncommon in present day hospital care to encounter specialists who demand treatment of injuries in their own field at the expense of other necessary treatment.

A neurological surgeon is often concerned only with the result of the injury to the central nervous system, and since his patients may die as a result of a head injury, he may consider a fracture as a relatively minor condition.

He may order delay in the treatment of a fracture on the grounds that an anesthesia, anoxia or an operative procedure on a bone might cause an aggravation of an intra-cranial lesion.

If, in his opinion, such other treatment might jeopardize the patient's life, he is quite justified in not permitting further treatment.

However, the neurological surgeon does not alway recognize that fractures need emergency treatment, and occasionally a bad result in a fracture is due to his unnecessary delay in treatment.

The same may be said for the chest surgeon and for the abdominal surgeon, although error in treatment is much less apt to occur as the result of an abdominal injury or chest injury as it is with a head injury.

If the responsible surgeon in charge of the multiple injury case has a broad knowledge of trauma, he will make sure that the patient receives proper treatment regardless of the specialty involved.

Summary

In view of the rapidly increasing number of multiple injuries as a result of auto crash injuries and the mass casualties that can be expected to arise in a world conflict, it is wise for each hospital to investigate its facilities for the care of a large number of seriously injured patients and organize a program based on the experiences of the minor catastrophes reported from various hospitals in this country.

The medical profession is responsible to educate the lay public and to help the Red Cross and other agencies develop plans to improve the first aid care in the case of catastrophes as well as auto crash injuries.

The care of the patient with multiple injuries in the accident room of most hospitals can be greatly improved. A concerted effort to fix the responsibility of the patient in the hands of one surgeon and to recognize the multiplicity of observations and treatment which are necessary to preserve life and limb and prevent disabilities, may result in improving our care of these patients.

Malaria in The Southwest

By JACK A. BERNARD, M.D., F.A.C.P. *and*
BRANCH CRAIGE, M.D., F.A.C.P., *El Paso*

Malaria is so rarely seen in the El Paso area that it is often unsuspected as a cause of fever. The observation of three cases in patients shortly after their return from a trip to Mexico prompts us to draw attention to malaria as a cause of undiagnosed febrile illness.

Case Reports

Case 1. J.A.S. was a 28-year-old male with a history of chills and fever daily for four days. He had been on a camping trip to Mexico two weeks before and had been to Guadalajara, San Blas and Mazatlán.

Physical examination revealed no abnormalities. The neck was supple. The chest was clear. The spleen was not palpable. Neurological signs were negative. The blood count showed 4.12 million red cells, hemoglobin of 12.3 grams and a white count of 6,800 with 40 percent polymorphonuclear leucocytes, 2 percent stab forms, 58 percent lymphocytes. A blood smear showed numerous malarial parasites.

The patient was placed on chloroquine and primaquine, and in 24 hours his temperature was normal. Except for weakness, he had no further symptoms and had a smooth recovery.

Case Two

E.P.W., a 38-year-old male, had had chills and high fever daily for five days and was treated with penicillin and other antibiotics with no improvement.

The diagnosis of malaria was not suspected until it was learned that the patient's friend (Case 1) had malaria and had been a companion on the above camping trip to Mexico.

Physical examination was negative except that the spleen was palpable one finger's breadth below the costal margin. The laboratory findings showed a red blood cell count of 3.47 million, hemoglobin of 14.25 grams and a white blood cell count of 3,050 with 51 percent polymorphonuclear leucocytes, 36 percent lymphocytes, and 8 percent stab forms. Malarial parasites were numerous in the blood smear.

The patient was placed on chloroquine and primaquine, responded promptly, and had no further difficulty.

Case Three

L.M., a 25-year-old housewife, had been in Mexico for one month and had visited Acapulco, Mexico City, Puebla, Taxco and Cuernavaca.

For eight days she had been ill with fever, headache and some stiffness of the neck.

She had been treated with penicillin, erythromycin and mixed sulfonamides for several days without relief. The temperature was 106°, but the physical examination was otherwise not remarkable. There were no neurological signs. Laboratory studies showed a blood count of 4.25 million red cells, 11.7 grams of hemoglobin (83 percent), 5,000 white cells, with 82 percent polymorphonuclear leucocytes, 12 percent lymphocytes, 5 percent monocytes and 1 eosinophile. A few malarial parasites in the schizont stage were seen on the blood smear. With chloroquine diphosphate recovery was rapid.

Diagnosis

The diagnosis of malaria is suggested in an otherwise obscure febrile illness by the following:

History of Travel in Endemic Malarial Areas. The incubation period of *Vivax* (tertian) malaria is 10 to 20 days or longer, depending upon the strain of *Vivax*. In recurrences the interval between attacks varies with the strain, the amount of immunity developed by the host, and the type of drug used to suppress the previous attacks.[1] In *Falciparum* malaria, symptoms may be of any variety; and a history of travel in the tropics may be the only suggestive clue.

Spiking Fever. The first few days of *Vivax* malaria may show an irregular remittent fever. It is very common later to have daily spikes due to a double tertian infection rather than fever every other day.[2] Brief high fever (105-106°) is commonly seen in malaria and there is probably no other disease in which a patient may look so healthy a few hours after such a temperature. Prostration and severe malaise is often lacking between severe febrile episodes.

Herpes Labialis commonly occurs in malaria as it does also in other infections especially the common cold, pneumococcal, and meningococcal diseases. Splenic enlargement, anemia, and cachexia

are late signs and should not be awaited before the diagnosis is suspected.

Diagnosis Established

Diagnosis is established by the observation of the malarial parasites in the thick or thin blood smear. Parasites may be overlooked early in the course of the disease because in the first attack of malaria there may be considerable fever before there is a very heavy parasitemia.[3]

The untreated patient gradually develops a heavier parasitemia and the demonstration of the parasites day by day becomes easier in such cases. In subsequent attacks because of increasing host resistance parasitemia becomes heavier before fever is produced and diagnosis accordingly becomes easier.[3] It is not necessary to wait for the chill or fever to take a blood specimen for examination.

On the other hand, the administration of even a small dose of suppressive drug may so reduce the number of visible parasites as to render diagnosis by blood smear impossible.

Accordingly, blood smears should be made before any antimalarial drug is given. Mild suppressive effect may result from various other drugs and make the diagnosis more difficult without relieving the patient's symptoms.

Treatment

Treatment of *Vivax* malaria is now simple, safe and reliable. In doubtful cases the use of chloroquine may be relied on to exclude the disease.

Chloroquine[4] is to be preferred over atabrine and quinine because of its prompter effect, longer suppressive action, freedom from staining the skin and relative lack of other toxic effects. The dosage is ten 0.25 gram tablets of chloroquine diphosphate (Aralen®) administered six the first day, in divided doses, and two on each of the next two days.

Fever and parasitemia will be eradicated in 48 to 72 hours or less; but the exoerythrocytic forms are not eliminated and relapse may occur in some patients at a later date.

The liability to relapse depends upon the strain of *Vivax,* the development of immunity, and the strength of the original inoculum. Amodiaquin (Camoquin®) is another suppressive drug which may be substituted for chloroquin.

Prophylaxis

Prevention of subsequent relapse may be assured by the use of primaquine, one 15 milligram (primaquine base) tablet daily for 14 days. Because a rare patient may develop hemolytic anemia, the blood and urine, particularly in colored individuals who are more prone to have this complication, should be observed for evidence of hemolysis and the drug stopped if any hemolysis is noted.

Primaquine is usually nontoxic in a dose of 15 milligrams a day for fourteen days and will destroy the exoerythrocytic forms. The exoerythrocytic forms are responsible for the continuing asymptomatic infection which in some patients results in periodic flareups of parasitemia and fever.

Summary

The world's most common serious infection is a rarity in the arid Southwest. Malaria should be suspected in febrile patients particularly in those returned from endemic areas.

The authors are indebted to Dr. Carl Milchen for permission to report case 2.

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Southwestern Association Lays Plans for October Meeting

The social program will be expanded and more time will be provided for questioning guest speakers on individual cases at the next annual meeting of the Southwestern Medical Association, scheduled for sunny Tucson October 23, 24, and 25.

According to Dr. Blair W. Saylor of Tucson, publicity chairman for the meeting, the Southwestern program will be unique in several aspects.

Small groups will be formed to enable the doctors to ask questions of the guest speakers regarding individual cases. The doctors will be able to circulate from one group to another, if they so desire. They are asked to bring along individual case histories.

The program will be arranged to provide more time for physicians to become acquainted and at the same time to maintain a high quality of scientific material. Slogan for the meeting will be "Learn and Have Fun Together".

MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

January 16, 1958

F. P. Bornstein, M. D., Editor—Case Number 958

Presentation of Case by C. P. C. Logsdon, M.D.

History—Dr. Nathan Kleban:

A 50-year-old unmarried Negro woman entered the hospital because of troubled breathing, on November 23, 1957, and died 20 hours later.

In February, 1952, the patient was seen for the first time with an illness consisting of sharp middle and left anterior chest pain which limited inspiration, cough, sputum, fever up to 104.6 for seven weeks, signs of left pleural effusion and an increase in size of the heart shadow on X-ray film.

There was moderate anemia; repeated blood tests showed positive Kolmer, negative Kahn; acid-fast organisms were not found in studies of four sputum specimens. She was treated for congestive heart failure and given anti-bacterial and antibiotic drugs.

For one week, three million units of penicillin were given daily. This was doubled during a second week, then stopped. After almost six weeks, the patient refused treatment and tests, was discharged with daily temperature elevations to 100, disappearance of the pleural effusion and decrease in heart size.

From Cardiac Clinic

The patient was admitted for the second time in February, 1955, referred from cardiac clinic because of left anterior chest pain when breathing, and swelling of the feet.

Hypothyroidism was suggested by low voltage of the QRS complexes in the limb leads of the electrocardiogram, but the protein-bound iodine determination was 6.0 gamma per cent.

The patient signed herself out of the hospital in one week. Pedal edema was unchanged. Breath sounds were distant and rales were present at the left base. The liver was not enlarged. A systolic murmur was described at the apex.

Chills, feverishness, breathlessness and mild left chest and right leg pain brought the patient to the hospital in October, 1955. There was pulmonary congestion, bilateral pleural effusion, no remarkable heart findings except enlargement, and probable pericardial effusion suggested by the roentgeneologist, and pedal edema. The liver was not felt.

Temperature elevation of 102 subsided to normal over a period of one week. Three sputum specimens were cultured for acid-fast bacilli with no growth. She was treated with a cardiac regime, antibiotics, and anticoagulants for thrombophlebitis. After 12 days, she signed herself out.

Occlusion Demonstrated

A venogram on January 21, 1957, demonstrated occlusion of the right greater, and of the left small, saphenous veins. Three days after the venogram, the patient entered for the fifth time, on January 24, 1957, with consolidation of the right middle lobe, which cleared in one week. A skin rash disappeared when penicillin-streptomycin injections were stopped. The February, 1955, history stated that the patient was allergic to penicillin.

Acute pyelonephritis brought the patient to the hospital in April, 1957. Penicillin and streptomycin were given for two days. Except for initial hypotension, there was a relatively uneventful recovery. Serological test for syphilis was negative.

Her seventh admission was in May-June, 1956. In addition to a previously recorded history of pneumonia in 1947, there had been a left hip and pelvic fracture sustained in an automobile accident in 1950, for which she was hospitalized at Torrance, California, General Hospital. There was no history suggesting rheumatic fever. Past history, system review, and family history were not otherwise pertinent.

Blood Pressure Normal

The attending physician found a normal blood pressure; sinus tachycardia, edema up to the level of the eighth thoracic verterba, distended neck veins; rales, diminished breath sounds and a percussion note which was duller at the left base than the right; a diffuse gentle heaving over the apical region of the pericardium, equal second aortic and pulmonic sounds, slight muffling of heart tones, grade I systolic mitral murmur, duplication of first mitral sound; liver edge five cm. below the right costal margin; hepatic jugular reflex; no myxedematous appearance.

Low voltage persisted on electrocardiograms; P-R interval was .21; P-wave was broad and

notched. Radioactive iodine uptake study was reported as "hypothyroidism secondary to pituitary dysfunction." BMR was minus 6 and cholesterol 170 mg. percent. Cardiac fluroscopy was reported as "no significant change." Diuresis, from 187½ to 134½ pounds occurred over a six weeks period.

The attending staff doctor favored a diagnosis of constrictive pericarditis. The cardiac consultant disagreed. He thought there was a grade II mitral and possible grade I secondary aortic systolic murmur. Discharge diagnosis was probable rheumatic heart disease.

The patient was re-admitted in July, 1957. She weighed 162 pounds on the sixth day; 147 when discharged six weeks later. Atrial fibrillation appeared and was converted to sinus rhythm by quinidine. Twenty micrograms of l-triiodothyronine daily was prescribed.

Cough productive of white sputum, increasing dyspnea, orthopnea and dependent edema hospitalized the patient for two weeks in October, 1957. There was an 11-pound diuresis from admission to the fifth day. She was no longer on thyroid hormone.

The tenth and final entry was on November 23, 1957, when the patient was breathing with great effort, complaining of pains in the swollen legs, and of weakness. She became hypotensive, and went into coma before she died.

Dr. Vincent Ravel:

The admission film shows a marked enlargement of the cardiac silhouette. There is, at this time, no evidence of congestive failure. The aorta is not particularly prominent.

I don't see any notching of the ribs or anything that would suggest a coarctation of the aorta, but a very prominent left ventricular segment suggests a hypertensive cardiovascular disease.

The next film dates from 1952, at which time the chest shows lobar type pneumonia in the right lower lobe, and there is a significant decrease in the size of the cardiac silhouette. This, of course, suggests a number of interesting possibilities.

The fact that the cardiac silhouette has decreased in size suggests that there may have been a pericardial effusion, or there may have been a myxedema with a subsequent re-establishment of the thyroid balance which would give more tone to the cardiac muscle.

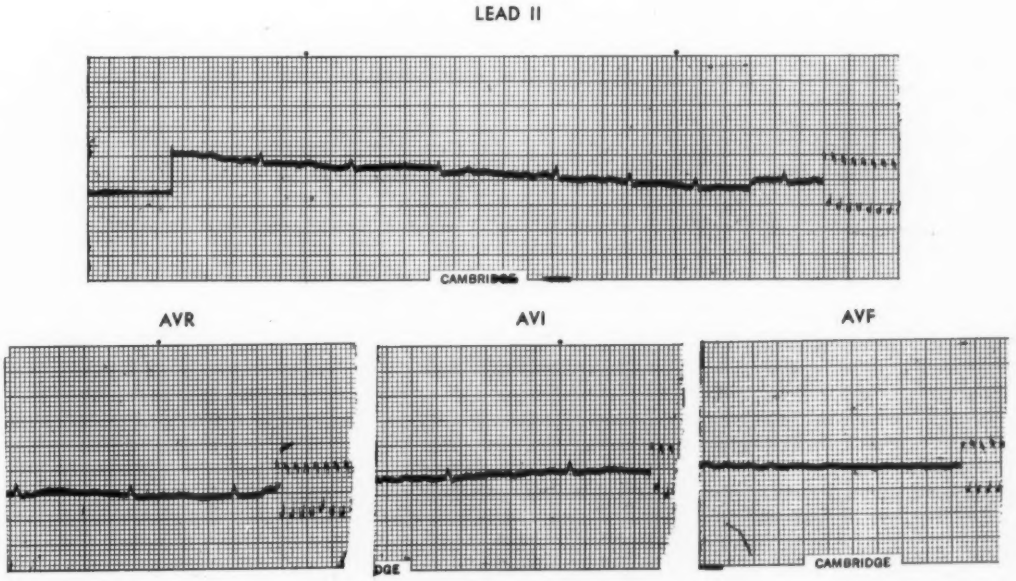
Next Film

The next film shows partial resolution and there is an interlobar collection of fluid in the chest. Then, in 1956, the patient again returned with a pneumonitis, this time in the right middle lobe, and a pleural effusion on the left.

Here again, we run into the problem of a myxedema with a hypothyroidism. The cardiac silhouette at this time, however, shows no significant change from the previous examination.

We did a venogram, and it showed occlusion of the saphenous on the right and the small saphenous on the left. I don't think those are particularly contributive, except as part of a peripheral failure syndrome.

As I understand, she had pitting edema of the



7/29/57—Atrial fibrillation.

extremities and peripheral failure. Again, we come back to the problem of a hypothyroidism.

Another Bout

Then we again observe another bout of pneumonitis and resolution. The film in 1957 shows a complete resolution of the pneumonitis at the right.

In chest radiography, whenever there are repeated attacks of lobar type pneumonia, you must consider a neoplasm or some sort of obstructive syndrome in the bronchial tree.

We have here bouts of pneumonitis in the right lower and right middle lobe.

These might be explained on the basis of adenoma in the bronchus.

I want to show three more films, of July of 1957, with all the classic findings of congestive failure with pleural effusion and the increased markings at the bases.

Then, following treatment with thyroid and diuretics, the cardiac silhouette keeps changing. Certainly we can't exclude a pericardial effusion or pericarditis.

It was pointed out in the examination of 1955 that this could be a pericarditis with effusion. The final film, in October, 1957, her last admission, shows the findings of congestive failure with pneumonitis.

Iodine Study

Now the radio-opaque iodine study that we did suggested a possible pituitary hypothyroidism secondary to pituitary dysfunction.

We were able to increase the function of the thyroid gland on its radio-active iodine up-take study which showed this was a secondary hypothyroidism due to pituitary dysfunction.

I think of possibly several explanations for this syndrome: (1) I think there is a hypothyroidism; (2) possibly a pericarditis; (3) possible bronchogenic adenoma or neoplasm which caused repeated attacks of pneumonitis in the right middle and right lower lobes.

Dr. Kleban:

I would like to know if we have any information about this patient between 1952 and 1955? She was repeatedly in the cardiac clinic but we don't have the clinical portions of the charts—only the hospital chart.

We have a KUB that shows opaque material in the buttocks, possibly anti-luetic therapy, and there again, an aortitis, a luetic aortitis or pericarditis has to be considered.

Dr. Logsdon:

I want to thank Dr. Kleban for the protocol, for it is a truly comprehensive record. It is almost impossible to include everything, although it seems that everything pertinent is included.

I believe it was in 1956 that the patient's status as a constrictive pericarditis came up again for consideration. It had always been suggested by her EKG.

Her EKG was characterized by extremely low voltage which would suggest either an extreme hypothyroidism or a constrictive pericarditis. However, at that time the diagnosis of constrictive pericarditis was apparently ruled out by the following three things:

(1) In constrictive pericarditis, the heart is a small heart. In the first place, it is usually caused by tuberculosis which does not attack the valves and myocardium and therefore does not cause a cardiomegaly. Any cardiomegaly and valvulopathy in constrictive pericarditis would be coincidental. It does not usually fit.

(2) Electrocardiographically, in constrictive pericarditis, the heart is fixed and cannot be moved and the same potential is recovered with the same leads. As I recall, when we moved her the potential shifted.

(3) There were murmurs and at times they seemed fairly significant, although not of anything particular. We could not pin them down to definite rheumatic heart disease, but we usually do not find murmurs in constrictive pericarditis.

Extensive Study

At that time, a rather extensive study was done by Dr. Kleban on the patient, and these studies showed hypothyroidism, following which the patient was controlled better than she had ever been before.

But I would like to point out for the first time she also came to the clinic regularly.

At that time, she was put on moderate doses of cytomel and I believe that she only tolerated 10 or 20 micrograms which is a pretty small dose, and she was given regular injections of mercury and she took diamox and digitoxin.

During that time, she was reasonably well controlled.

I think something happened that precluded the possibility of any real control of the patient's edema, and that was an intractable right heart failure. It may or may not have had something to do with myxedema. I don't see exactly how it could have.

Heart Failure

Apparently intractable right heart failure existed and we could never bring the patient under too good a control. Subsequently, she developed a very severe sensitivity to mercury and from that time on her course was progressively downhill.

The only time after that she was significantly free of edema was when tubes were put in her and left for quite a long time. Over a period of weeks, she drained down to practically normal.

Following that, she was never completely free of edema and free of discomfort, and we never had her under control again.

So I feel that the edema was due more to severe right heart failure than to any possible myxedema.

That diagnosis was made on the basis of iodine take-up, but the PBI was normal.

First Illness

About the first illness, the protocol states that she was sent for the first time for an illness consistent with sub-acute bacterial endocarditis with a left anterior chest pain, limited respiration, cough, sputum, fever up to 104.

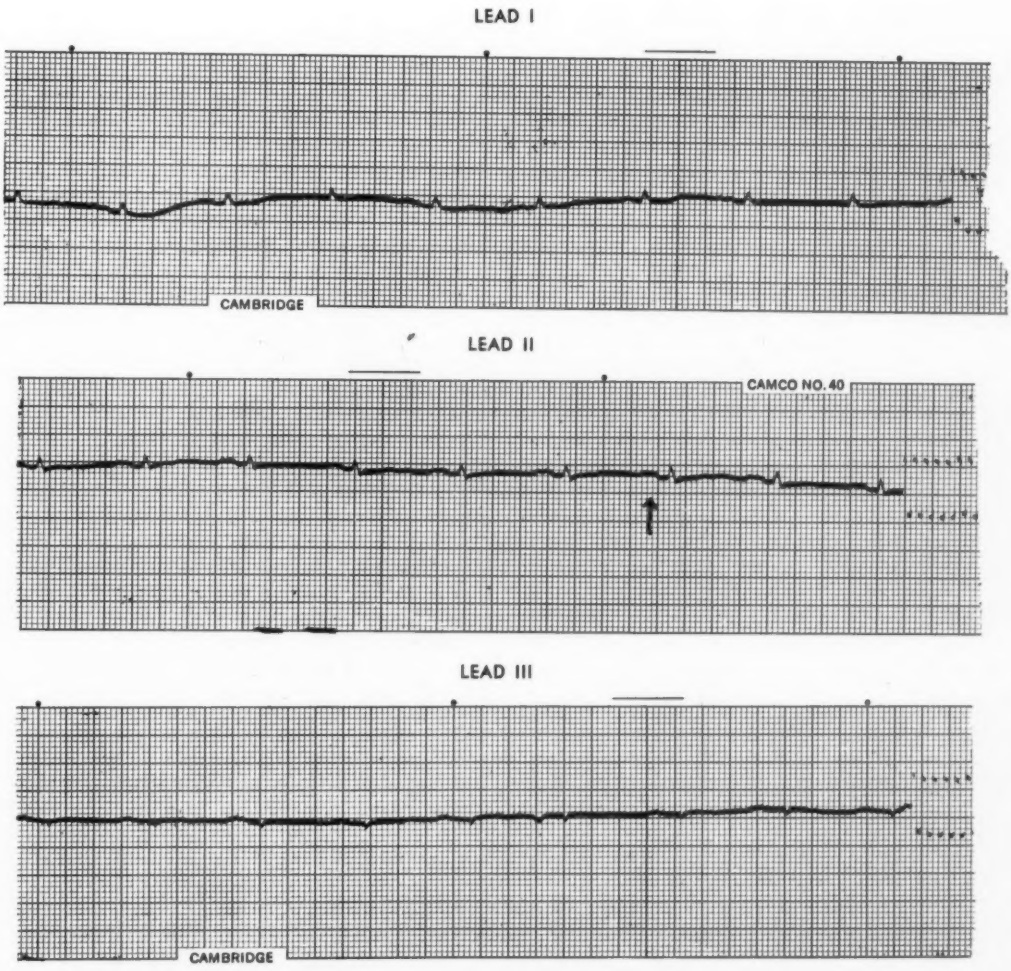
In the differential diagnosis, I will consider only the things that we considered at the time. I won't attempt an encyclopedic differential diagnosis.

1. It became fairly apparent that the patient had a severe right heart failure and there was no adequate explanation apparent for the cause of it.

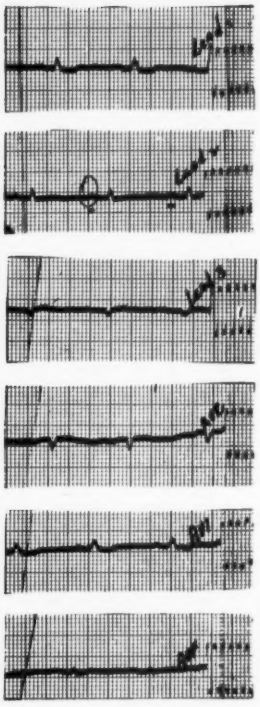
2. We considered very seriously rheumatic heart disease, but you would have to postulate some fairly silent lesions.

I would say possibly the last year and a half of her life, she had a grade II at least, maybe grade III, systolic murmur at the apical area.

Pershaps we didn't hear it constantly, but



8/3/57—Return of normal sinus rhythm. EKG suggests left atrial enlargement.



10/18/57—First degree AV block.

we heard it every now and then. It was a wholly systolic murmur. It was not, however, the type of murmur that you would expect to hear in a significant mitral regurgitation.

There were no pathognomic signs of mitral stenosis. So, if we are dealing with rheumatic fever, we would have to postulate first that this was rheumatic fever in 1952.

The febrile illness that she had in 1952 did not respond to treatment and I don't remember that illness personally.

At that time, she had heart damage and then she developed silent lesions, perhaps a mitral regurgitation, and we would still have to postulate a precocious and fairly intense pulmonary vascular resistance developing to give us a subsequent cor pulmonale type of right heart failure which ultimately became intractable.

Definite Hypertension

There is something that was not mentioned on the protocol. In fact, it may not have appeared in our hospital chart. At times, she had a very definite hypertension, but not a severe one, always moderate.

Added to whatever may have been the cause of her right heart failure could have been a myxedema, although it did not appear to us that myxedema played a very important part, at least in the last few years of her illness, which were the only years that we witnessed.

Now, another thing that might have determined the character of her later illness, and which was pointed out by Dr. Ravel, is pulmonary embolization. Some of the pulmonary symptoms here might have been due to repeated pulmonary embolization.

I would like to suggest the possibility of right heart failure due to repeated pulmonary embolization with occlusion of the multiple radicles of the pulmonary arterial tree.

I think, terminally I would expect a rather larger heart than that.

She must have had a good deal of thrombosis

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up and down the venous system up to and including the right auricle, but aside from those possibilities, I can think of nothing very significant.

That is, right heart failure with a possible etiology of pulmonary embolization, possible silent rheumatic heart disease.

Dr. Bornstein:

In a cardiac silhouette, is there any way to differentiate between the outline of the atria and the ventricles?

Dr. Ravel:

Yes, the atria can be differentiated by oblique views. The anatomist really got things a little confused in describing the heart, because what we are seeing in a PA view of the chest is really an oblique view of the heart. In order to get a PA view of the heart, and a lateral view of the heart, you have to put the patient in both obliques. In a saggital view of the chest here, you are seeing an oblique view of the heart. In order to differentiate the various chambers, you have to put the patient in the oblique position and then you get true saggital and lateral views of the heart.

Clinical Diagnosis: Intractable right heart failure with elephantiasis.

Dr. Logsdon's Diagnosis: 1. Right heart failure and pulmonary embolization. **2.** Silent rheumatic heart disease.

Pathological Diagnosis: 1. Chronic constrictive pericarditis. **2.** Chronic dilatation of both atria. **3.** Extreme peripheral edema.

Pathological Discussion—Dr. F. P. Bornstein:

On autopsy, we found the body of a middle aged, well nourished Negro woman. The most prominent feature on external examination was the extreme edema of the lower extremities which had swollen to proportions of elephantiasis.

The skin over the extremeties was hard and indurated.

The pleural cavities were obliterated by adhesions.

The pericardial cavity was completely obliterated by stone-hard fibrous adhesions.

These adhesions made the examination of the heart somewhat difficult because the heart could not be shelled out of these adhesions.

The adhesions were such that they formed a constrictive band between the atria and the ventricles which produced a very extreme dilatation of the atria.

In a way, then, this was a heart which was

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suffering from a combined mitral and tricuspid stenosis, inasmuch as the entire valvular ring was constricted by external pressure.

The ventricles were small, the coronary arteries were not remarkable.

In addition, there was acute pulmonary edema.

On microscopic examination, it was interesting to note that while the superficial layer of the pericardium was composed of scar tissue, an intensive, active inflammatory process was present in the deeper layers.

This inflammatory process extended into the superficial layers of the myocardium. In short, this was not simply a mechanically impaired heart, but a heart which still was suffering from an active chronic inflammatory process.

I am unable to come to any precise conclusion about the nature of this inflammatory process. It is somewhat suggestive of a rheumatic type inflammation, but this is only a suggestion.

The thyroid was not remarkable. The peripheral venous system was free of thromboses.

Summary

In summary, then, we have a woman with a long standing history of heart disease and decompensation. The main difficulty was a mechanical one due to a constrictive pericarditis which was still actively progressing at the time of death.

Coming Meetings

Texas State Surgical Society, El Paso, Texas, April 5-8, 1958.

Southwestern New Mexico Medical Society, Rio Mimbres Country Club, Deming, N. M., Apr. 9, 1958.

Texas Medical Association, Annual Meeting, Houston, April 18-21, 1958. Annual Meeting of the Texas Orthopaedic Association will be held in conjunction with meeting of Texas Medical Association.

Fifth International Congress of Internal Medicine, Sheraton Hotel, Philadelphia, April 24-26, 1958.

Arizona Medical Association, Inc., Annual Meeting, Chandler, Ariz., April 30 - May 3, 1958.

New Mexico Medical Society, Annual Meeting, Albuquerque, N. M., May 14-16, 1958.

American Fracture Association, annual meeting, Oklahoma City, Oct. 1-3, 1958.

Southwestern Medical Association, annual meeting, Tucson, Oct. 23-25, 1958.

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